**Dissertation On**

# "THE EFFECTS OF HYPER VENTILATION IN LAPAROSCOPIC SURGERIES ON HAEMODYNAMICS , PARTIAL PRESSURE OF CARBONDIOXIDE AND ACID BASE STATUS"

**Submitted to the**

**TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY**

**in partial fulfillment of the requirements**

**for the award of degree of**

**M D (BRANCH X)**

**ANAESTHESIOLOGY**


**STANLEY MEDICAL COLLEGE**

**THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY**

**CHENNAI ,TAMILNADU**

**MARCH -2009**

# CERTIFICATE

This is to certify that this dissertation entitled **"EFFECTS OF HYPERVENTILATION ON HAEMODYNAMICS, PARTIAL PRESSURE OF CARBONDIOXIDE AND ACID BASE STATUS"** is a bonafide original work of **Dr.B.GAYATHRI** in partial fulfillment of requirement for **M.D. Anaesthesiology** Examination of **The Tamil Nadu Dr.M.G.R.Medical University** in March 2009.

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# DECLARATION

I, **Dr.B.GAYATHRI,** solemnly declare that the Dissertation Titled **"EFFECTS OF HYPERVENTILATION ON HAEMODYNAMICS, PARTIAL PRESSURE OF CARBONDIOXIDE AND ACID BASE STATUS"** is a bonafide work done by me at Govt. Stanley Medical College Hospital during the period Aug. 2007 to March 2008 under the expert guidance and Supervision of **Dr.P.CHANDRASEKAR, M.D., D.A.,** Professor & HOD, Dept. of Anaesthesiology, Govt. Stanley Medical College, Chennai-1.

This dissertation is submitted to **The Tamil Nadu Dr.M.G.R.Medical University** in partial fulfillment of requirement for the award of **M.D.Degree Anaesthesiology.**

Place:

Date: **(Dr.B.GAYATHRI)**

# ACKNOWLEDGEMENT

I wish to express my sincere thanks to **Prof. Dr.MOHANA SUNDARAM, M.D, Ph.D**, Dean, Government Stanley Medical College and Hospital for having kindly permitted me to utilize the facilities of the hospital for the conduct of the study.

My heartfelt thanks to **Prof. Dr. P.CHANDRASEKAR M.D, D.A**, Prof and HOD, Department of ANAESTHESIOLOGY, Govt. Stanley Medical College and Hospital for his motivation, valuable suggestions, constant supervision and for all necessary arrangements for conducting the study .

I am greatly indebted to **Prof.Dr.B.KALA M.D, D.A, Prof.Dr.R.MADHANKUMAR M.D, D.A , and Prof.Dr.S.GUNASEKARAN M.D, D.N.B,** for their guidance throughout the study.

I am immensely thankful to D**R.SATISH LOGIDASAN M.D, D.N.B** for his valuable guidance in performing and consolidation of the study.

I thank all the **ASSISTANT PROFESSORS** who evinced keen interest and gave support without which this study would not have been possible.

I thank **Mr. PADMANABHAN,** Statistician, for helping me in the statistical analysis. I thank all the postgraduates for their valuable support during the study period.I thank all theatre personnel for their co-operation.

I thank all the patients without whose participation, this study would not have been possible.

# CONTENTS

# INTRODUCTION

Surgical procedures have been improved to reduce trauma to the patient morbidity, mortality and hospital stay with consequent reductions in health care costs.

Laparoscopy for clinical purposes was first performed by Jacobaeus in 1910, and, following advances in optical and lighting systems and the work of Steptoe (1965, 1969)

The provision of better equipment and facilities along with increased knowledge and understanding of anatomy and pathology, has allowed the development of endoscopy for diagnostic and operative procedures.

In early 1970s, various pathologic gynecologic conditions were diagnosed and treated using laparoscopy. This endoscopic approach was extended to cholecystectomy in late 1980s. Nowadays laparoscopy is used in colonic, gastric, splenic, hepatic and urologic surgeries.

Although laparoscopic surgeries are minimally invasive, uptake of C02 from the pressurized pneumo peritoneum can cause clinically relevant hypercarbia and respiratory acidosis with physiological consequences. Hypercarbia and acidosis occurs due to absorption via huge peritoneal cavity, decreased lung compliance and insufficient ventilation . C02 is 20 times more soluble/diffusible than oxygen which is

insufflated in pressurized form (10-15 mmHg). If duration of surgery is prolonged, systemic absorption of C02 will be more and it requires aggressive hyperventilation.

The pneumoperitoneum and the patient positions required for laparoscopy induce pathophysiologic changes that complicate anaesthetic management.

In this study I am studying the effects of hyperventilation on haemodynamics, partial pressure of carbon dioxide and acid base status in laparoscopic cholecystectomy.

This study was conducted in the Department of Anesthesiology, Stanley medical college hospital, Chennai. The period of study is August 2007 to March 2008.

# AIM OF THE STUDY

The aim of the study is to compare the effects of hyperventilation with 3 different respiratory rates and tidal volume of 10 ml /kg on haemodynamics like mean arterial pressure, pulse rate and partial pressure of carbon dioxide and acid base status in laparoscopic cholecystectomy.

## VENTILATORY AND RESPIRATORY CHANGES DURING LAPAROSCOPY

### 1.VENTILATORY CHANGES

Pneumo peritoneum decreases thoracic compliance by 30 – 50 % in healthy, obese, and ASA class III or IV patients but the shape of pressure volume loop does not change after the pneumo peritoneum is created and kept constant. Compliance is not affected by subsequent patient tilting or by increasing the minute ventilation required to avoid intra operative hypercapnia.(4)

Reduction in functional residual capacity due to elevation of diaphragm and changes in the distribution of pulmonary ventilation and perfusion from increased airway pressure can be expected. However, increasing IAP to 14mmHg with the patient in a 10 -20 degree head up or down position does not significantly modify physiologic dead space or shunt in patients without cardiovascular problems.(21)

## 2. INCREASE IN PARTIAL PRESSURE OF ARTERIAL CARBONDIOXIDE.

1. Absorption of carbon di oxide from the peritoneal cavity .
2. VA/Q mismatch: increased physiological dead space
   Abdominal distension
   Position of the patient.
   Controlled mechanical ventilation.
   Reduced cardiac output.

   These mechanisms are accentuated in sick patients (e.g obesity ASA II or III)
3. Increased metabolism ( insufficient plane of anaesthesia)
4. Depression of ventilation by anaesthetics
   (e.g spontaneous breathing)
5. Accidental events
   CO2 emphysema
   Capnothorax
   Co2 embolism.(21)

During uneventful CO2 pneumo peritoneum, the increase in partial pressure of arterial carbondioxide progressively increases to reach a plateau 15 to 30 minutes after beginning of CO2 insufflation in patients under

controlled mechanical ventilation during gynaecologic laparoscopy in Trendelenburg position or laparoscopic cholecystectomy in head –up position.(22)

Any significant increase in Paco2 after this period requires a search for a cause independent of or related to co2 insufflation ,such as co2 subcutaneous emphysema. The increase in paco2 is dependent on intra abdominal pressure.

Because it takes 15 to 30 minutes for Paco2 to plateau, anaesthetic techniques using spontaneous breathing should be limited to short procedures at low IAPs.

Capnography and pulse oximetry provide reliable monitoring of Paco2 and arterial oxygen saturation in healthy patients and in the absence of acute intra operative disturbances.

Although mean gradients between Paco2 and the end tidal carbon dioxide tension PEtCO2 do not change significantly during peritoneal insufflation of CO2 ,individual patient data regularly show variations of this difference during pneumo peritoneum .

Paco2 and Δa-$ETCO_2$ increase more in ASA class II and III patients than ASA class I patients. These finding have been documented in patients with chronic obstructive disease patients and in children with cyanotic congenital heart disease. These data therefore highlight the lack of correlation between $PaCO_2$ and $PEtCO_2$ in sick patients particularly with impaired $CO_2$ excretion capacity, and in otherwise healthy patients with acute cardiopulmonary disturbances.(2)

Consequently, arterial blood sampling is recommended when hypercapnia is clinically suspected, even in the absence of abnormal $PEtCO_2$. Post operative intra abdominal $CO_2$ retention results in increased respiratory rate and $PCO_2$ and $PETCO_2$ of patients breathing spontaneously after laparoscopic cholecystectomy as compared with open cholecystectomy.(23)

The observation of an increase in $PaCO_2$ when $CO_2$, but not nitrous oxide or helium was used as the insufflating gas suggests that the main mechanism of increased $PaCO_2$ during $CO_2$ pneumo peritoneum is absorption of $CO_2$ rather than the mechanical ventilatory repercussions of increased IAP. Accordingly direct measurement of $CO_2$ elimination using

metabolic monitor combined with investigation of gas exchange showed a 20 % TO 30% increase of $VCO_2$ without significant changes in physiologic dead space in healthy patients undergoing pelvic laparoscopy (IAP of 12 to 14 mmHg)in the head down position or laparoscopic choloecystectomy in the head up position.(26)

The absorption of a gas from the peritoneal cavity depends on

1. Its diffusibility.
2. The absorption area .
3. The perfusion of the walls of the cavity.

The limited rise of $PaCO_2$ actually observed can be explained by the capacity of the body to store $CO_2$ and by impaired perfusion due to increased IAP.(8)

Although increased Paco2 may be well tolerated by young, otherwise healthy patients, the extent to which hypercapnia is acceptable has not been determined and probably varies according to the patient's physical status. It is wise to maintain $Paco_2$ within physiologic ranges by adjusting controlled mechanical ventilation. Except in special circumstances such as $Co_2$ subcutaneous emphysema, correction of increased $Paco_2$ can be easily achieved by a 10%-25% increase in alveolar ventilation. (16)

## CAPNOGRAPHY END TIDAL CARBON DI OXIDE MONITORING

Capnography is the graphic record of instantaneous carbon dioxide concentrations in the respired gases during a respiratory cycle.

The primary goal of anesthesiologist is to prevent hypoxia .While pulse oxymeter is a direct monitor of oxygenation, Capnography is an indirect monitor. It is a noninvasive technique that can monitor $PaCO_2$. In normal individual the arterial –End tidal $PCO_2$ may vary from 2-5mmHg. The $PETCO_2$ is even more useful if its relationship to $PaCO_2$ can be established initially by blood gas analysis. Thereafter, changes in $PaCO_2$ may be assumed to occur in parallel with those in $PETCO_2$.

The (a-ET) $PCO_2$ is a measure of alveolar dead space, and changes in alveolar dead space correlate well with changes in (a-ET) $PCO_2$. So (a-ET) $PCO_2$ is an indirect estimate of V/Q mismatching of the lung.

Changes in body position, temperature and pulmonary blood flow ,as well as mechanical ventilation and cardiopulmonary bypass, can result in changes in the ventilation perfusion status of the lungs .This in turn alters the alveolar dead space fraction and the slope of phase III ,and this affects (a-ET)PCO2 .Further there is no consistent correlation between (a-ET)PCO2 and the various factors mentioned.

**HAEMODYNAMIC PROBLEMS IN LAPAROSCOPIC SURGERIES**.

**[Cardiac output, Mean Arterial Pressure, Pulse Rate]**

An increase in intra abdominal pressure causes reflex increase in vagal tone due to excessive stretching of the peritoneum may produce bradycardia. The threshold pressure that has minimal effects on haemodynamic function is less than 12 mm of Hg. A biphasic change in the cardiac output is observed .Up to an IAP of 10 mm of Hg the cardiac filling pressures are normal or increased and Cardiac output improves.(24)

But if the inflation pressures are increased further more than 15mm of Hg the insufflated $CO_2$ compresses both the venous capacitance and the arterial resistance vessels. This produces a rise in the systemic vascular resistance (SVR), and the pulmonary vascular resistance leading to an increased after load.(10)

The mechanism of the decrease of cardiac out put is multifactorial. A decrease in venous return is observed after a transient increase in venous return seen at low IAP (< 10mm Hg).

# Increased IAP results in

1. Caval compression,
2. Pooling of blood in the legs,
3. and an increase in venous resistance.

The decline in venous return, which parallels the decrease in cardiac output, is confirmed by a reduction in left ventricular end diastolic volume measured using trans esophageal echo cardiography. The reduction in venous return and cardiac output can be attenuated by increasing circulating volume before the pneumoperitoneum.

Mean arterial blood pressure rises and the cardiac output falls (25-35%). An IAP more than 20 mm of Hg reduces the renal and mesenteric blood flow markedly. As IAP rises cardiac output falls. The combined effect of anaesthesia head –up tilt and peritoneal insufflation can reduce cardiac index by 50 %.(7)

These haemodynamic changes are well tolerated by healthy individuals, but may have deleterious consequences in patients with cardiovascular disease.

The increase in systemic vascular resistance is affected by patient position. Whereas the Trendelenburg position attenuates this increase, the head-up position aggravates it. The patient's circulating volume affects changes in venous return and changes in after load. The increase in

systemic vascular resistance can be corrected by administration of vasodilating anesthetic agents, such as isoflurane, or direct vasodilating drugs, such as nitroglycerin or nicardipine.(27)

The increase in systemic vascular resistance is considered to be mediated by mechanical and neurohumoral factors. The return of hemodynamic variables to baseline is gradual and takes several minutes, suggesting the involvement of neurohumoral factors. Catecholamines, the renin-angiotensin system, and especially vasopressin are all released during pneumoperitoneum and may contribute to increasing afterload.(28)

> The increase in systemic vascular resistance also explains why the arterial pressure increases, whereas the cardiac output falls.(28)

# REVIEW OF LITERATURE

1. Wittgen CM et al. Analysis of hemodynamic and ventilatory effects of laparoscopic cholecystectomy. Archives of surgery 1991; 126:997-1001. Studied 20 patients with normal pre op cardio pulmonary status and 10 patients who had previously diagnosed cardiac pulmonary disease. Demographic, haemodynamic, arterial blood gas and ventilatory data were collected before peritoneal insufflation and at intervals during surgery. During $CO_2$ insufflation significant decreases in arterial pH values and significant increases in $PaCO_2$ occurred in group 2 patients compared to group 1 .The peak inspiratory pressures did not increase significantly.

2. Bhavani Shankar et al Arterial to End –tidal Carbon Dioxide Pressure difference during laparoscopic surgery in pregnancy Anaesthesiology 2000, 93:370-3 found out that capnography is adequate to guide ventilation during laparoscopic surgery in pregnant patients. Respiratory acidosis did not occur when $PEtCO_2$ was maintained at 32 mm of Hg during carbon dioxide pneumo peritoneum.

3. Juraj Sprung et al, Anaesthesia analgesia 2003; 97:268 -74 Studied the effects of morbid obesity, 20 mm Hg of pneumo peritoneum and body posture ( 30 deg head down and 30 deg head up) They concluded that the alveolar – arterial difference in oxygen tension was not affected by body position, pneumo peritoneum or the mode of ventilation.

4. Sprung J, Whalley G, Falcone T et al. The impact of morbid obesity, pneumoperitoneum, and posture on respiratory system mechanics and oxygenation during laparoscopy. Anaesthesia analgesia 2002;94 :1345-50. Respiratory mechanics during laparoscopy was affected by pneumo peritoneum and obesity but not by change in position.

5. Maharjhan S K Shrestha et al. Kathmandu university medical journal 2007 studied the effects of hyperventilation in 60 patients undergoing laparoscopic cholecystectomy .they divided the patients into 2 groups .first group received 12/minute ventilation with a tidal volume of 10ml/kg and the second group received 15/minute ventilation with the same tidal volume . They observed that there was no significant changes in the mean arterial pressure but there

was a significant increase in $PCO_2$ and $ETCO_2$ and a significant decrease in pH in control group than the study group. They found that 10 -15 % increase in minute volume is beneficial during $CO_2$ pneumo peritoneum to prevent the adverse effects of hypercarbia and acidosis.

6. Hideo iwasaka MD , Hiroshi miyakawa et al Canadian journal of anaesthesia 1996 found that Peak inspiratory pressure increased and there was a decrease in pH and accumulation of carbon dioxide with peritoneal insufflation.

7. T.Kazama, K .Ikeda British journal of anaesthesia 1996 found that during carbon dioxide pneumo peritoneum carbon dioxide output increased by 49%.

8. K. Kubota ,N. Kajiura Surgical endoscopy 1993 ,studied the respiratory function and haemodynamics in patients undergoing laparoscopic cholecystectomy by doing ABG analysis and Swan ganz catheter for cardiac function monitoring . They found that there was a significant increase in PCO2 and a significant decrease in pH , base excess after creation of pneumo peritoneum when

compared to the values measured before institution of pneumo peritoneum . But there was no change in cardiac output , pulmonary arterial pressure or pulmonary arterial wedge pressure.

9. Mrinmoy das et al, Indian journal of anaesthesia, 2007, sixty patients randomly allocated to 2 groups to receive T. Clonidine or control. Studied the effects of oral clonidine premedication in haemodynamics of patients undergoing laparoscopic surgeries and found that there was a significant rise in pulse rate ,systolic blood pressure , diastolic blood pressure and mean arterial pressure during pneumo peritoneum.

10. K. Dhoste et al, Canadian journal of anaesthesia 1996, studied the effects of laparoscopic cholecystectomy on respiratory function and haemodynamics in elderly ASA III patients. Haemodynamic variables were measured before and after induction and 15 ,30 ,60 minutes after peritoneal insufflation . They found that there was a significant increase in cardiac index ,pulse rate, systolic , diastolic blood pressures and mean arterial pressure and there was a significant increase in PCO2 in laparoscopic surgeries after 15 minutes of pneumo peritoneum.

11. Y.S. Kim et al The catholic university of Seoul ,Korea ,European journal of anaesthesiology , 2008 ,found that arterial PCO2 should be checked intermittently in prolonged pneumo peritoneum during laparoscopic colorectal surgery to confirm adequate ventilation.

12. Baratz RA et al , Anaesthesiology ,1969, Blood gas studies during laparoscopic surgeries under general anaesthesia .With adequate controlled ventilation PCO2 did not rise significantly during CO2 insufflation in laparoscopic surgeries.

13. Smith , Benzie British journal of Anaesthesiology ,1971, investigated the cardiovascular effects of peritoneal insufflation of carbondioxide found that there was a significant increase in airway pressure ,intra thoracic pressure, central venous pressure and signs of cardiac stimulation like increase in pulse rate and blood pressure. End tidal carbon dioxide rose only slightly.

# MATERIALS AND METHODS

Ninety patients of ASA physical status 1 undergoing elective laparoscopic cholecystectomy lasting a minimum of 45 minutes.

Patients belonging to the age group of 20 – 60 years of both sexes

It is a prospective randomized study .The study was approved by Institutional ethical committee and written informed consent was obtained from patients.

## INCLUSION CRITERIA

1. ASA physical status I.
2. Patients undergoing laparoscopic cholecystectomy lasting more than 45 minutes.

# EXCLUSION CRITERIA

1. Patients suffering from any respiratory diseases (bronchial asthma, chronic bronchitis, emphysema and respiratory failure)
2. Congestive cardiac failure
3. Renal failure.

# MATERIALS

Ninety patients of both sexes undergoing laparoscopic cholecystectomy were randomly allocated into three groups .

Group 1 – ventilated with rate of 12 /min and tidal volume of 10 ml/kg

Group 2- ventilated with rate of 14 /min and tidal volume of 10 ml/kg.

Group 3 – ventilated with rate of 16/min and tidal volume of 10ml/kg.

Drugs to be kept ready for anaesthesia

1. Inj.Glycopyrrolate 0.2 mg ampoules,
2. Inj. Fentanyl 50 micg /ml ampoules,
3. Inj. Propofol vials 1% ,
4. Inj. Succinyl choline hydrochloride vials,
5. Inj. Atracurium ampoules ,
6. Inj. Neostigmine ampoules.
7. Isoflurane
8. Appropriate size endotracheal tubes.

9. Drager Fabius anaesthesia machine.
10. Multiparameter monitor with Electro cardiogram ,pulse oximetry ,end tidal carbondioxide monitoring and non invasive blood pressure.
11. 20 G IV cannula for radial artery cannulation.
12. 2ml heparinised plastic syringes, flask and ice for transportation of ABG sample.

## PREPARATION OF THE PATIENT

Patients were advised overnight fasting.

All patients were given T. Ranitidine 150 mg , T.Diazepam5mg on the previous night of surgery and on the morning of surgery .

All the patients were pre medicated with Inj. Glycopyrrolate 10mics/ kg IM 45 minutes before surgery.

After shifting to the theatre right cephalic vein was cannulated with 18 G IV cannula and Ringer Lactate was started.

After attaching the monitors for electro cardiogram ,oxygen saturation and non invasive blood pressure basal parameters were recorded.

Patients were given Inj. Fentanyl 2 micg /kg for analgesia and induced with Inj. Propofol 2 mg /kg and paralysed with Inj.Succinyl choline 1.5mg/kg.

After adequate relaxation intubated with appropriate size endotracheal tubes and connected to DRAGER-FABIUS ventilator with the tidal volume of 10 ml/kg and ventilator rates as assigned to the patient. Maintained with N2O and O2 at 3 and1.5 litres per minute. And Isoflurane of o.4 to o.6%.

Patient's left radial artery was cannulated with 20 G IV cannula and connected to a three way adaptor and flushed with heparin saline to maintain the patency. An arterial sample was collected and sent for analysis.

Arterial Blood Gas analysis was sent 30 min after pneumoperitoneum and after exsufflation of $CO_2$.

Throughout surgery intra abdominal pressure was maintained at 12 cm $H_2O$.

Mean arterial pressure, Peak Inspiratory pressure, Heart rate, $EtCO_2$ were measured baseline and every 15 minutes thereafter. Increase in Mean arterial pressure of more than 20% controlled with Isoflurane. After the surgery is over and adequate respiratory attempts the patient was reversed with Inj. Neostigmine 50 micg /kg and Inj. Glycopyrrolate 10 micg/Kg dose . After the return of adequate muscle power and return of reflexes the patient was extubated after adequate oral suction.

## OBSERVATION AND RESULTS

The study was conducted in Stanley Medical College Hospital General Surgery operation theatres.

ASA Grade

All patients of three groups belonged to ASA grade I .

## DEMOGRAPHIC PROFILE

The sample of 90 patients was taken for the study .Data was expressed as mean +_SD or absolute values .Qualitative analysis was compared with **ANOVA TEST.**

The level of statistical significance was set at $p < 0.05\%$ .

The patient in each group was statistically comparable in distribution of age, weight and sex distribution.

The groups are

1. Group 1 consisting of 30 patients who were ventilated with a rate of 12 /minute with 10 ml /kg of tidal volume.

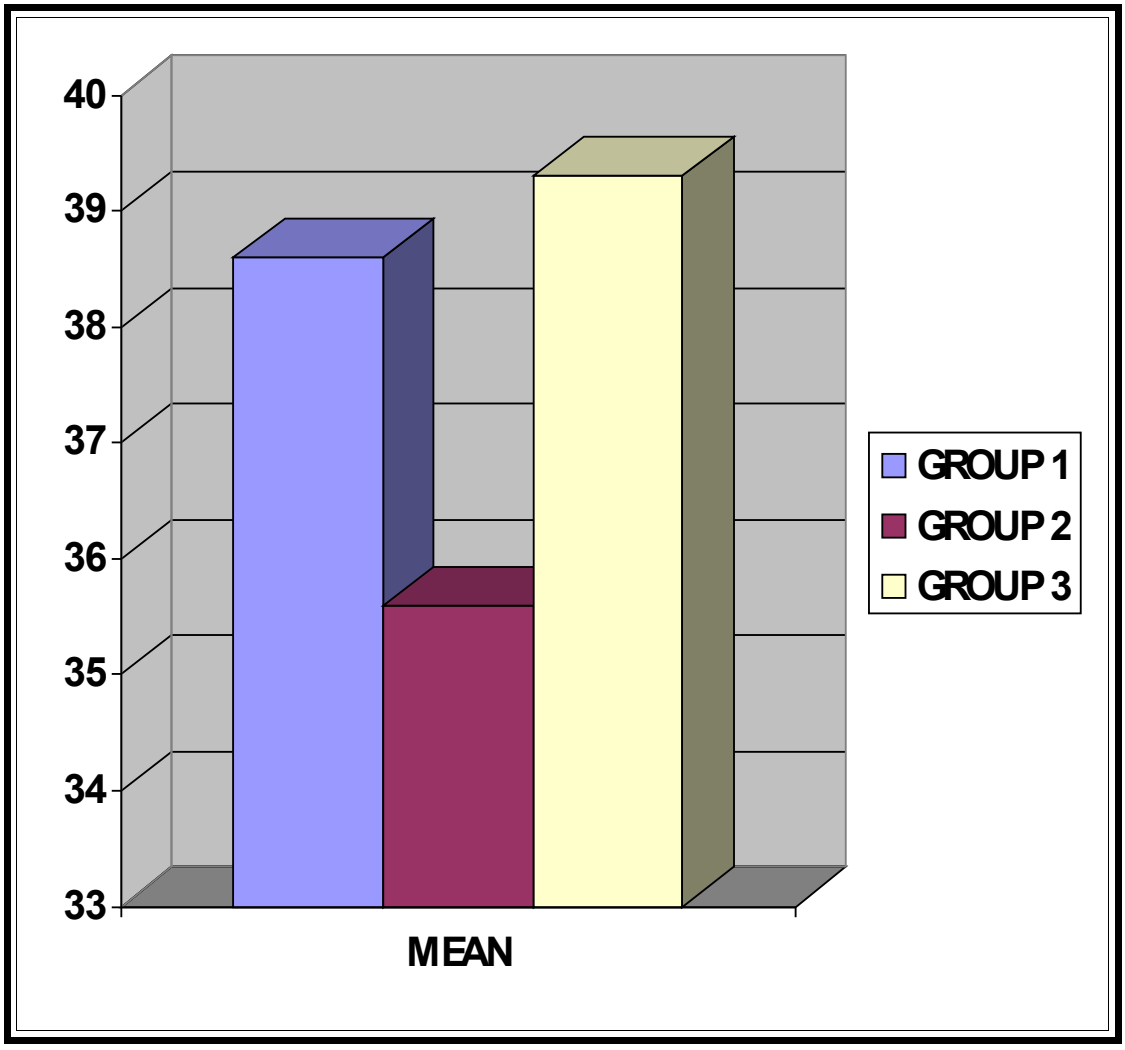
2. Group 2 consisting of 30 patients who were ventilated with a rate of 14 /minute with 10ml/kg of tidal volume.

3. Group 3 consisting of 30 patients who were ventilated with a rate of 16 /minute with 10 ml/kg of tidal volume.

# TABLE 1

## COMPARISON OF AGE GROUP

| GROUP | N | MEAN (yrs) | SD |
|---|---|---|---|
| 1 | 30 | 38.6 | 14.58 |
| 2 | 30 | 35.6 | 12.70 |
| 3 | 30 | 39.3 | 12.67 |
| TOTAL | 90 | 37.8 | 13.29 |
| | | P VALUE 0.527 | |
| | | NOT SIGNIFICANT | |

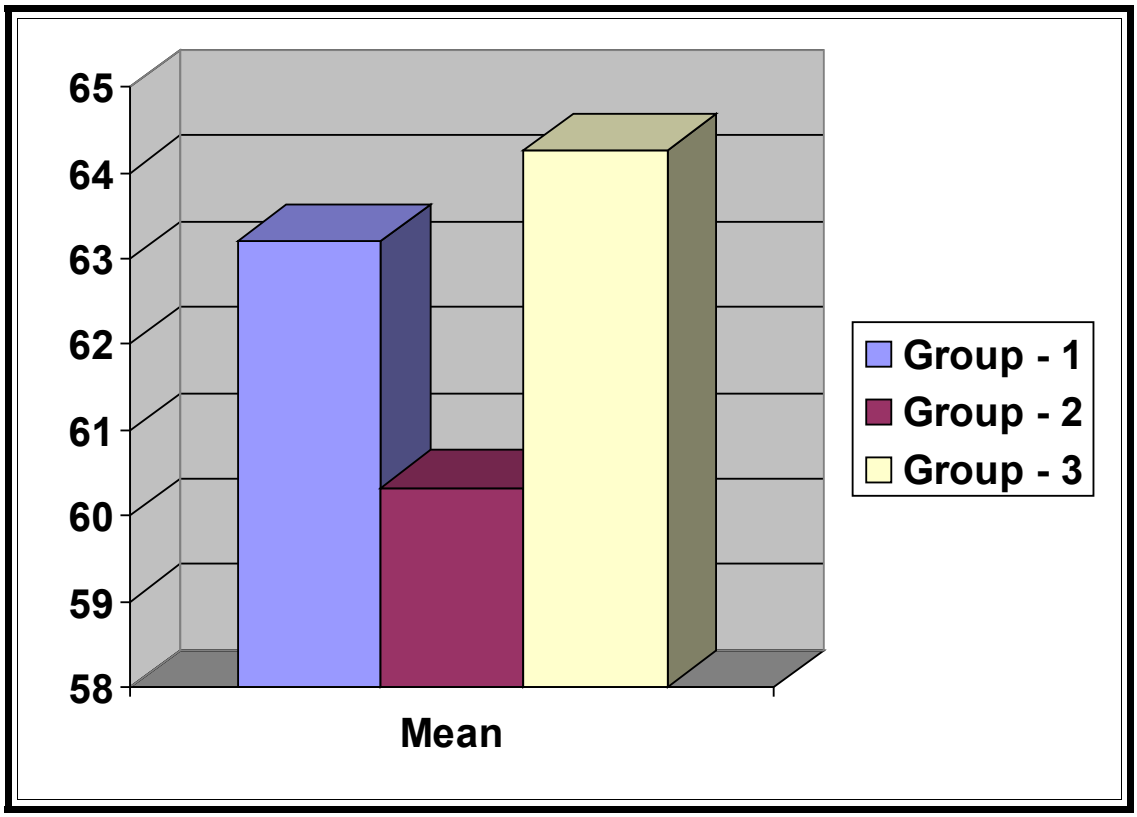



# TABLE 2

## COMPARISON OF WEIGHT

| | N | MEAN (Kg) | SD |
|---|---|---|---|

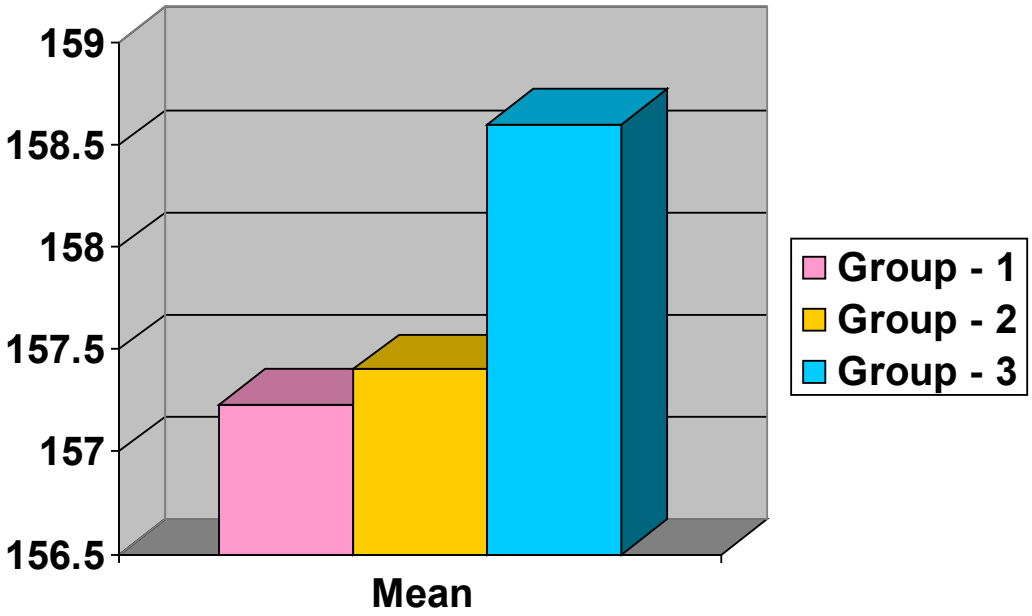
| GROUP | | | |
|---|---|---|---|
| 1 | 30 | 63.2 | 10.17 |
| 2 | 30 | 60.33 | 9.44 |
| 3 | 30 | 64.26 | 9.93 |
| TOTAL | 90 | 62.6 | 9.88 |
| | | P VALUE 0.248<br>NOT SIGNIFICANT | |



# TABLE 3

# COMPARISON OF HEIGHT

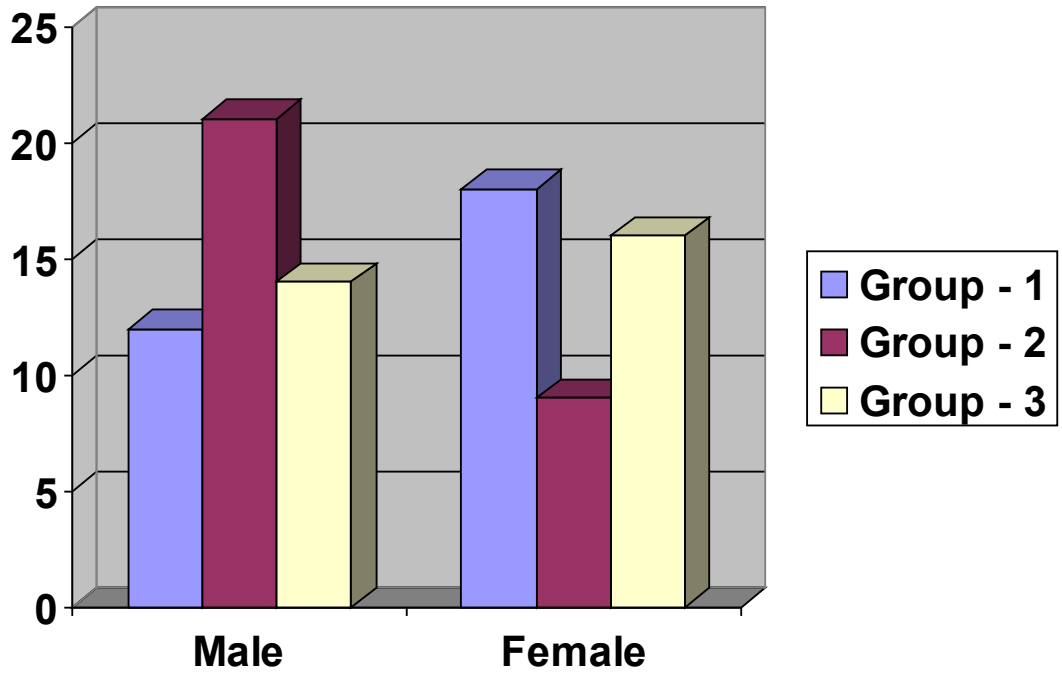
| Group | N | Mean (cm) | SD |
|---|---|---|---|
| 1 | 30 | 157.233 | 4.59 |
| 2 | 30 | 157.4 | 4.35 |
| 3 | 30 | 158.6 | 4.46 |
| Total | 90 | 157.744 | 4.46 |
| | | p value 0.438<br>NOT SIGNIFICANT | |



# TABLE 4

# COMPARISON OF SEX

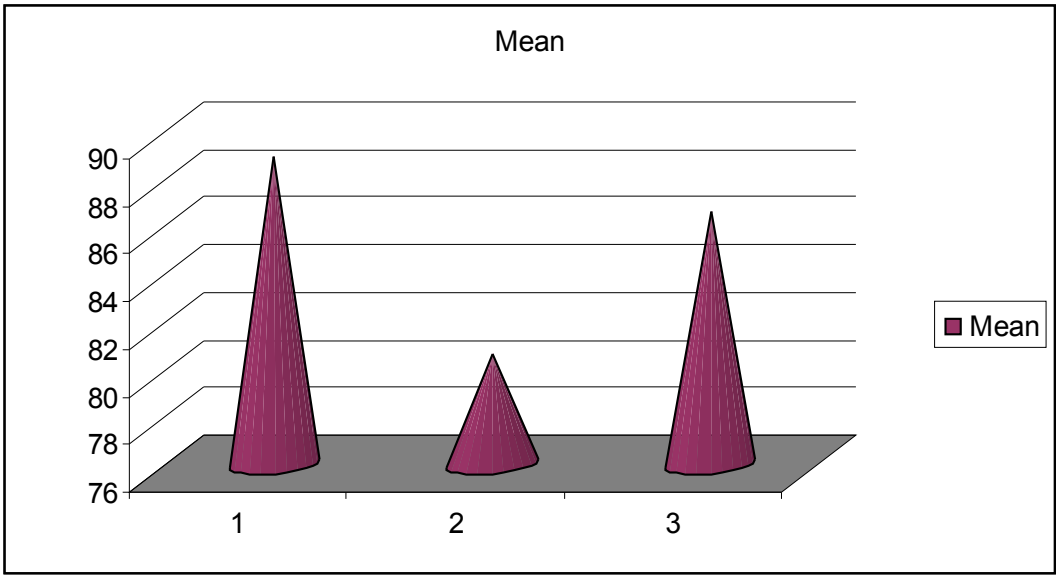
| | Group | | | |
|---|---|---|---|---|
| **Sex** | **1** | **2** | **3** | **Total** |
| **Male** | 12 | 21 | 14 | 47 |
| **Female** | 18 | 9 | 16 | 43 |
| **Total** | 30 | 30 | 30 | 90 |
| | | | p value 0.314<br>NOT SIGNIFICANT | |



# TABLE 5

## COMPARISON OF ANESTHESIA DURATION

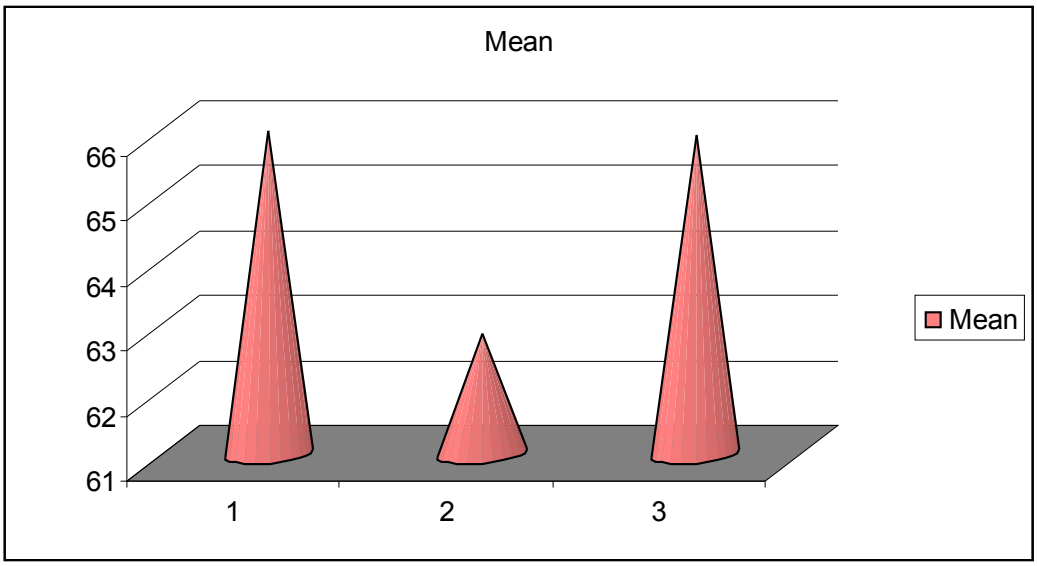
| Group | N | Mean (min) | SD |
|---|---|---|---|
| **1** | 30 | 88.86 | 18.04 |
| **2** | 30 | 80.6 | 12.05 |
| **3** | 30 | 86.56 | 14.13 |
| **Total** | 90 | 85.34 | 15.19 |
| | | p value 0.093<br>NOT SIGNIFICANT | |



# TABLE 6

# COMPARISON OF SURGERY DURATION

| Group | N | Mean (min) | SD |
|---|---|---|---|
| **1** | 30 | 65.96 | 11.19 |
| **2** | 30 | 62.83 | 7.17 |
| **3** | 30 | 65.9 | 9.14 |
| **Total** | 90 | 64.9 | 9.32 |
| | | p value 0.335<br>NOT SIGNIFICANT | |



# TABLE 7

## COMPARISON OF MEAN ARTERIAL PRESSURE

## PRE PNEUMOPERITONEUM

| Group | N | Mean( mm Hg) | SD |
|---|---|---|---|
| **1** | 30 | 84.6 | 9.74 |
| **2** | 30 | 86.7 | 8.85 |
| **3** | 30 | 83.63 | 8.92 |
| **Total** | 90 | 84.3 | 8.82 |
| | | p value 0.067<br>NOT SIGNIFICANT | |

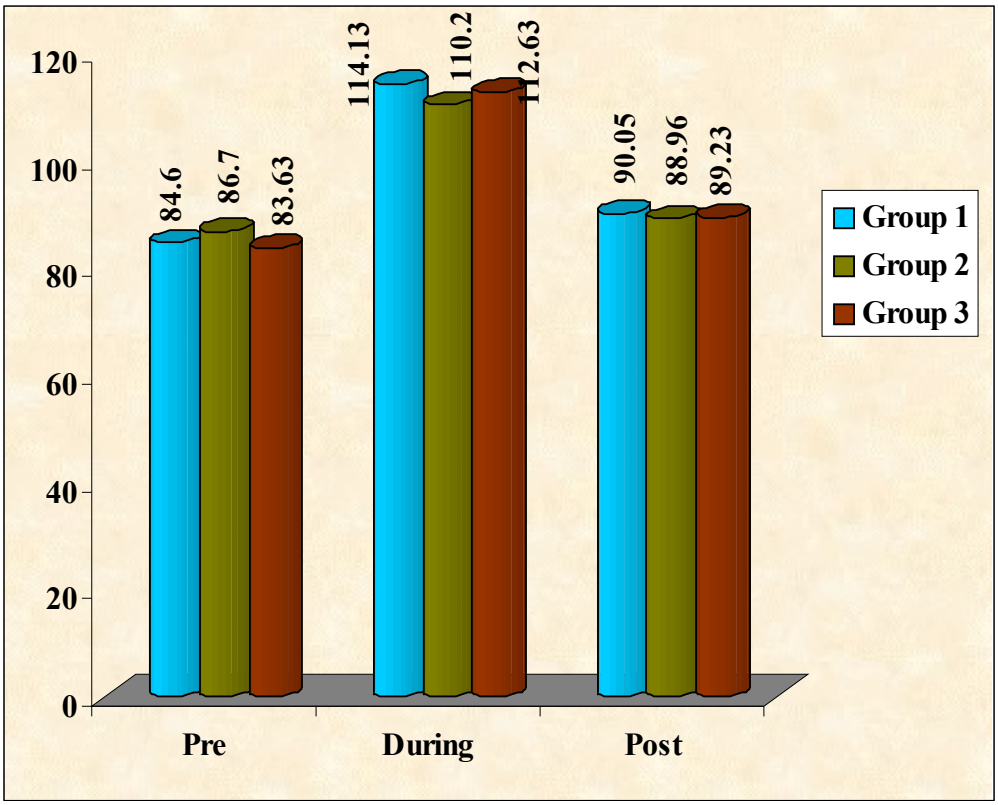
## DURING PNEUMO PERITONEUM

| Group | N | Mean( mm Hg) | SD |
|---|---|---|---|
| **1** | 30 | 114.13 | 16.57 |
| **2** | 30 | 110.2 | 8.03 |
| **3** | 30 | 112.63 | 9.42 |
| **Total** | 90 | 102.6 | 10.68 |
| | | p value 0.157<br>SIGNIFICANT | |

## POST PNEUMOPERITONEUM

| Group | N | Mean (mm Hg) | SD |
|---|---|---|---|
| 1 | 30 | 90.05 | 11.34 |
| 2 | 30 | 88.96 | 6.81 |
| 3 | 30 | 89.23 | 7.8 |
| Total | 90 | 89.3 | 8.76 |
| | | p value 0.142<br>SIGNIFICANT | |

## COMPARISON OF MEAN ARTERIAL PRESSURE




## COMPARISON OF pH

## PRE PNEMOPERITONEUM

| Group | N | Mean | SD |
|---|---|---|---|
| **1** | 30 | 7.4415 | 0.043 |
| **2** | 30 | 7.4516 | 0.0305 |
| **3** | 30 | 7.4505 | 0.051 |
| **Total** | 90 | 7.4393 | 0.045 |
| | | p value 0.073<br>NOT SIGNIFICANT | |

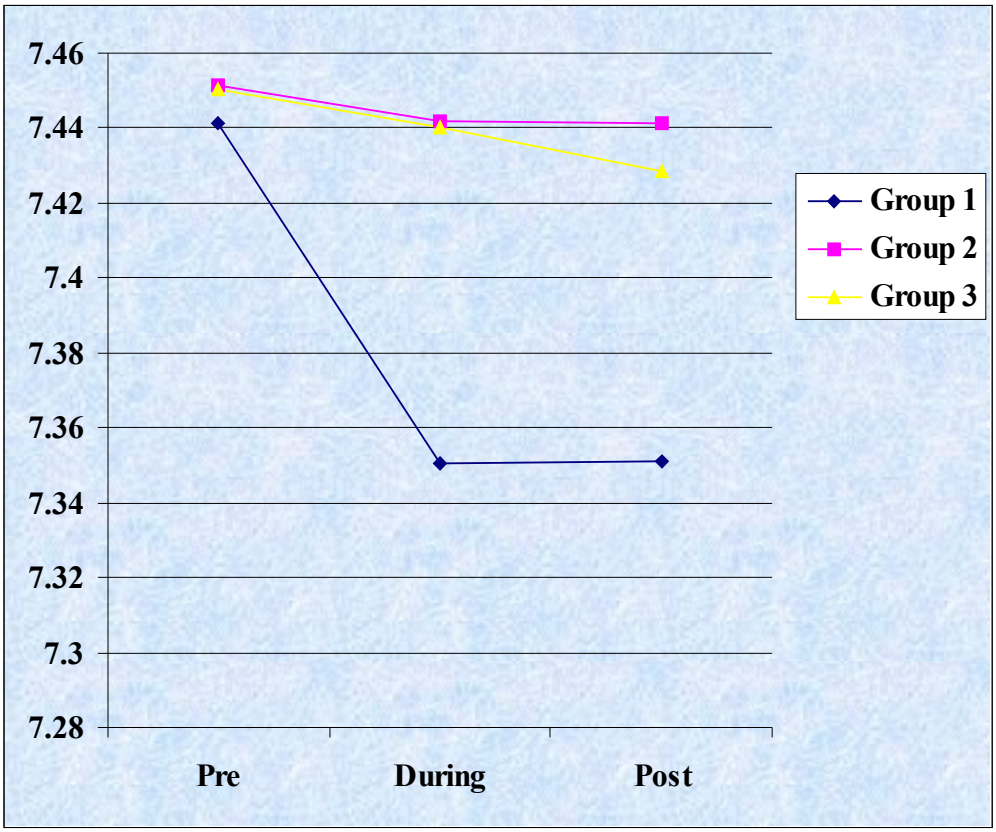
## DURING PNEUMOPERITONEUM

| Group | N | Mean | SD |
|---|---|---|---|
| **1** | 30 | 7.3505 | 0.0183 |
| **2** | 30 | 7.4416 | 0.0351 |
| **3** | 30 | 7.4405 | 0.0466 |
| **Total** | 90 | 7.4109 | 0.0553 |
| | | p value 0.000<br>SIGNIFICANT | |

## POST PNEUMOPERITONEUM

| Group | N | Mean | SD |
|---|---|---|---|
| **1** | 30 | 7.3511 | 0.0221 |
| **2** | 30 | 7.4413 | 0.0325 |
| **3** | 30 | 7.4283 | 0.0408 |
| **Total** | 90 | 7.4069 | 0.0515 |
| | | p value 0.000<br>SIGNIFICANT | |

## COMPARISON OF PH



# COMPARISON OF $PCO_2$

## PRE PNEUMOPERITONEUM

| Group | N | Mean( mm Hg) | SD |
|---|---|---|---|
| **1** | 30 | 39.0303 | 2.6863 |
| **2** | 30 | 34.9267 | 3.2585 |
| **3** | 30 | 33.1363 | 2.4725 |
| **Total** | 90 | 35.6978 | 3.7361 |
| | | p value 0.000<br>SIGNIFICANT | |

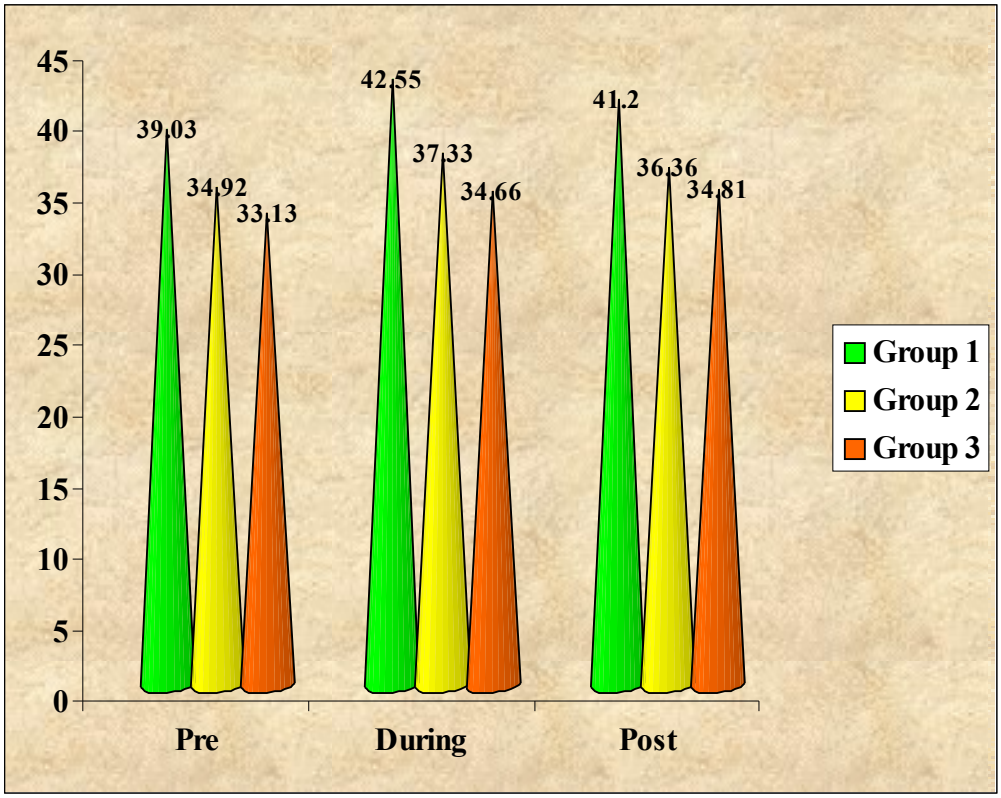
## DURING PNEUMOPERITONEUM

| Group | N | Mean( mmHg) | SD |
|---|---|---|---|
| **1** | 30 | 42.5547 | 2.9579 |
| **2** | 30 | 37.3367 | 3.5174 |
| **3** | 30 | 34.6687 | 3.8512 |
| **Total** | 90 | 38.1867 | 4.7499 |
| | | p value 0.000<br>SIGNIFICANT | |

## POST PNEUMOPERITONEUM

| Group | N | Mean (mmHg) | SD |
|---|---|---|---|
| 1 | 30 | 41.209 | 2.451 |
| 2 | 30 | 36.366 | 3.348 |
| 3 | 30 | 34.81 | 1.822 |
| Total | 90 | 37.4619 | 3.768 |
| | | p value 0.000<br>SIGNIFICANT | |

## COMPARISON OF $PCO_2$




## COMPARISON OF $ETCO_2$

## PRE PNEUMOPERITONEUM

| Group | N | Mean (mm Hg) | SD |
|---|---|---|---|
| **1** | 30 | 33.5 | 2.529 |
| **2** | 30 | 29 | 2.243 |
| **3** | 30 | 26.73 | 2.664 |
| **Total** | 90 | 29.74 | 3.746 |
| | | p value 0.000<br>SIGNIFICANT | |

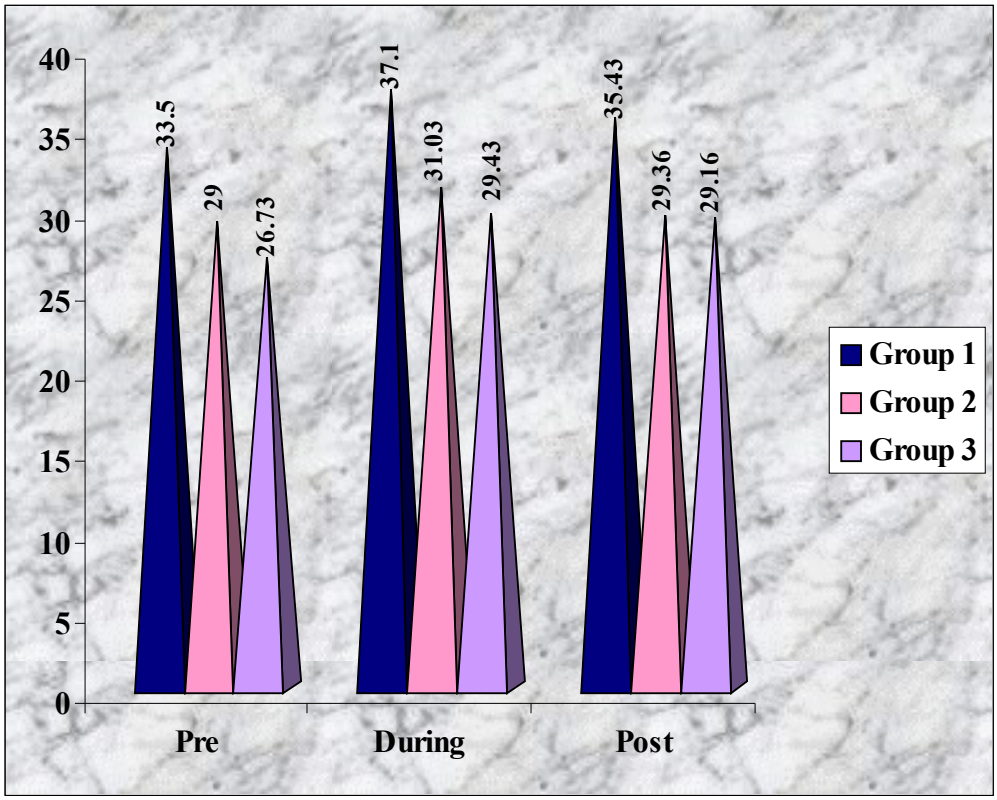
## DURING PNEUMOPERITONEUM

| Group | N | Mean (mmHg) | SD |
|---|---|---|---|
| **1** | 30 | 37.1 | 2.656 |
| **2** | 30 | 31.03 | 2.846 |
| **3** | 30 | 29.43 | 2.92 |
| **Total** | 90 | 32.52 | 4.329 |
| | | p value 0.000<br>SIGNIFICANT | |

## POST PNEUMOPERITONEUM

| Group | N | Mean( mm Hg) | SD |
|---|---|---|---|
| **1** | 30 | 35.43 | 2.83 |
| **2** | 30 | 29.36 | 2.83 |
| **3** | 30 | 29.16 | 2.98 |
| **Total** | 90 | 31.32 | 4.08 |
| | | p value 0.000<br>SIGNIFICANT | |

# COMPARISON OF $ETCO_2$



# COMPARISON OF BICARBONATE

## PRE PNEUMOPERITONEUM

| Group | N | Mean (mmol/l) | SD |
|---|---|---|---|
| **1** | 30 | 22.6 | 0.695 |
| **2** | 30 | 24.44 | 1.848 |
| **3** | 30 | 23.81 | 1.877 |
| **Total** | 90 | 23.61 | 1.734 |
| | | P VALUE 0.372<br>NOT SIGNIFICANT | |

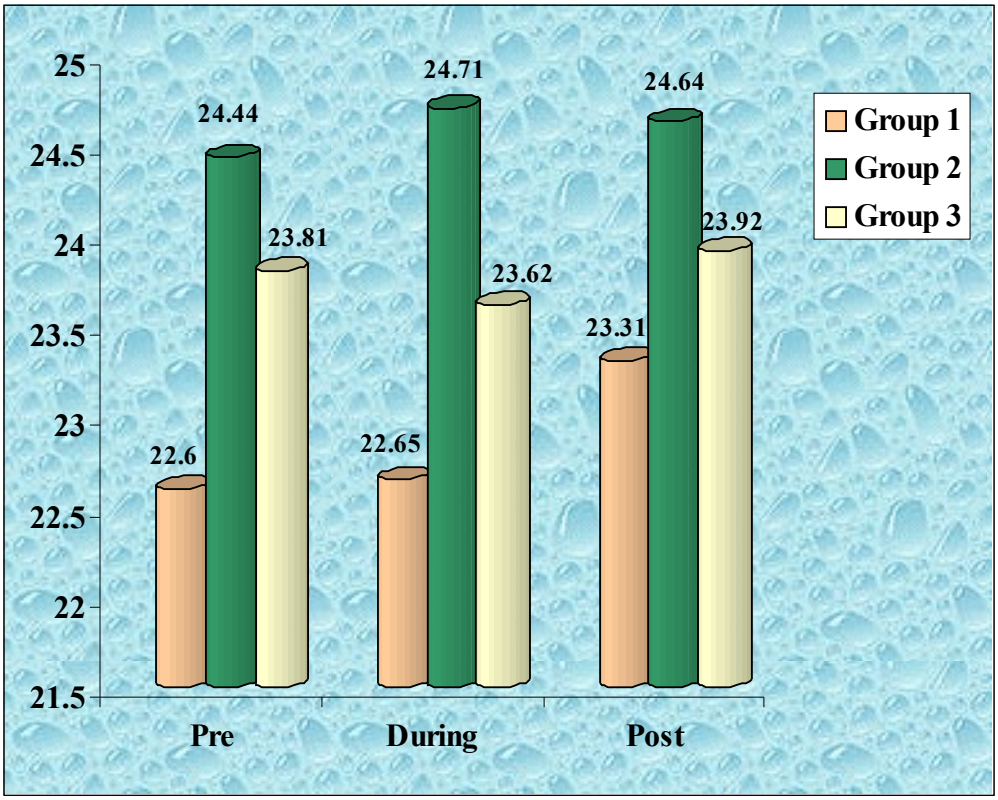
## DURING PNEUMOPERITONEUM

| Group | N | Mean ( mmol/l) | SD |
|---|---|---|---|
| **1** | 30 | 22.65 | 0.806 |
| **2** | 30 | 24.71 | 1.665 |
| **3** | 30 | 23.62 | 1.989 |
| **Total** | 90 | 23.66 | 1.767 |
| | | p value 0.590<br>NOT SIGNIFICANT | |

## POST PNEUMOPERITONEUM

| Group | N | Mean (mmol/l) | SD |
|---|---|---|---|
| 1 | 30 | 23.31 | 1.15 |
| 2 | 30 | 24.64 | 1.7 |
| 3 | 30 | 23.92 | 1.718 |
| Total | 90 | 23.95 | 1.62 |
| | | p value 0.970<br>NOT SIGNIFICANT | |

## COMPARISON OF BICARBONATE




## COMPARISON OF PULSE RATE

## PRE PNEUMOPERITONEUM

| Group | N | Mean | SD |
|---|---|---|---|
| **1** | 30 | 80.93 | 6.52 |
| **2** | 30 | 81.53 | 8.2 |
| **3** | 30 | 83.56 | 10.5 |
| **Total** | 90 | 82.47 | 8.9 |
| | | p value 0.143<br>NOT SIGNIFICANT | |

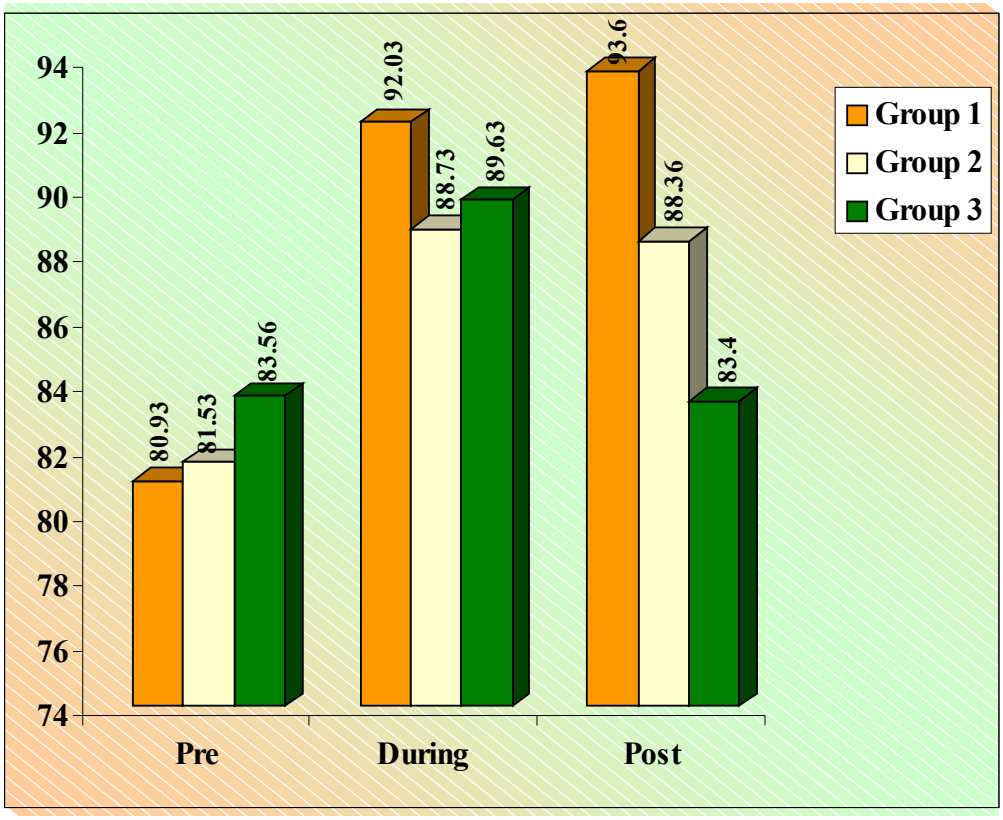
## DURING PNEUMOPERITONEUM

| Group | N | Mean | SD |
|---|---|---|---|
| **1** | 30 | 92.03 | 8.49 |
| **2** | 30 | 88.73 | 8.23 |
| **3** | 30 | 89.63 | 8.61 |
| **Total** | 90 | 90.13 | 8.46 |
| | | P VALUE 0.031<br>SIGNIFICANT | |

## POST PNEUMOPERITONEUM

| Group | N | Mean | SD |
|---|---|---|---|
| **1** | 30 | 93.6 | 8.04 |
| **2** | 30 | 88.36 | 6.19 |
| **3** | 30 | 83.4 | 6.51 |
| **Total** | 90 | 88.45 | 8.05 |
| | | p value 0.000<br>SIGNIFICANT | |

# COMPARISON OF PULSE RATE



# COMPARISON OF PEAK INSPIRATORY PRESSURE

# PRE PNEUMO PERITONEUM

| Group | N | Mean( cm of H2O) | SD |
|---|---|---|---|
| **1** | 30 | 20.7 | 1.89 |
| **2** | 30 | 20.6 | 1.92 |
| **3** | 30 | 20.63 | 1.56 |
| **Total** | 90 | 20.64 | 1.78 |
| | | P VALUE 0.976<br>NOT SIGNIFICANT | |

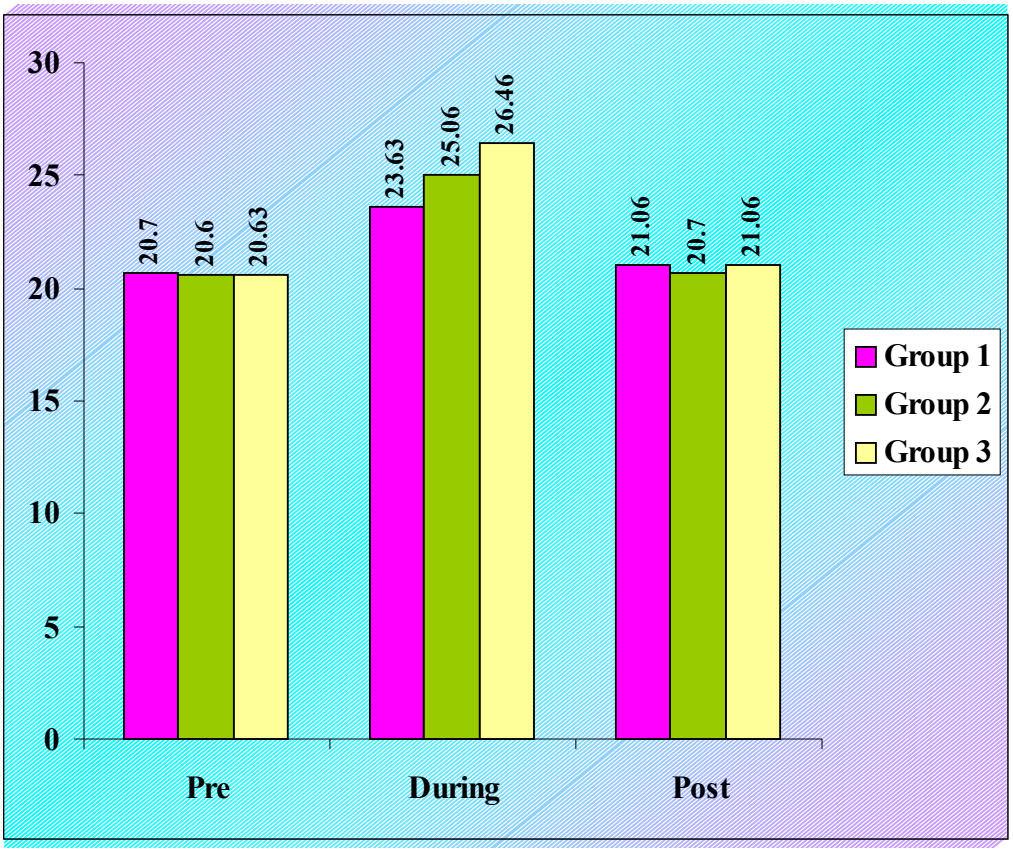
# DURING PNEUMO PERITONEUM

| Group | N | Mean (cm of H2O) | SD |
|---|---|---|---|
| **1** | 30 | 23.63 | 1.9 |
| **2** | 30 | 25.06 | 1.99 |
| **3** | 30 | 26.46 | 1.71 |
| **Total** | 90 | 25.05 | 2.18 |
| | | p value 0.000<br>SIGNIFICANT | |

## POST PNEUMOPERITONEUM

| Group | N | Mean (cm of H2O) | SD |
|---|---|---|---|
| 1 | 30 | 21.06 | 1.59 |
| 2 | 30 | 20.7 | 1.84 |
| 3 | 30 | 21.06 | 1.46 |
| Total | 90 | 20.94 | 1.63 |
| | | p value 0.608<br>NOT SIGNIFICANT | |

## COMPARISON OF PEAK INSPIRATORY PRESSURE




## DISCUSSION

During laparoscopic surgeries carbondioxide pneumoperitoneum is created and the effects of hypercarbia on the circulatory system are complex. This usually includes an increase in cardiac output, heart rate ,force of myocardial contraction ,blood pressure ,central venous pressure ,vasoconstriction in the pulmonary vessels and decreased peripheral resistance. Healthy ASA I patients are less likely than ASA III patients to undergo extreme changes.

General anaesthesia with intubation and mechanical ventilation results in a decrease in functional residual capacity which is caused by loss of muscle tone, diaphragmatic displacement and loss of thoracic volume .Lung compliance drops ,airway pressures increase and V/Q abnormalities occur. These changes are exaggerated by Trendelenburg position especially in elderly patients, obese and those with preexisting cardiopulmonary disease.

Various studies as mentioned in the review of literature have studied the effects of laparoscopy on haemodynamics and respiratory function.

This study correlates with the study done by Maharjan.K, Shreshtha et al ,which concludes that increasing the minute ventilation by 10 -15 % has beneficial effects during pneumoperitoneum to prevent hypercarbia

and acidosis

## MEAN ARTERIAL PRESSURE

There was no significant difference in mean arterial pressure before creation of pneumo peritoneum in all the three groups .(p value 0.067). During pneumoperitoneum there was an increase in mean arterial pressure in all the 3 groups but there was no significant difference in between the groups . ( p value 0.157) and the mean arterial pressure decreased after deflation of pneumoperitoneum but there was no significant difference in between the groups (p value 0.142).

## pH

pH did not show any significant difference between the study and control groups before creation of pneumo peritoneum.(p value 0.073). During pneumoperitoneum the pH varied significantly from control to study groups but no significant difference was observed between the study groups. After deflation also the control group differed significantly from study groups( p value 0.000 ). But no significant difference was observed between the study groups ( p value 0.127).

**PaCO2**

PaCO2 values measured before pneumoperitoneum showed a higher value in group 1 whereas it was in lower normal range in groups 2 and 3. The values are 34.92 mm of Hg and 33.13 mm of Hg respectively. It was statistically significant and the p value is 0.000.

In between groups 2 and 3 there was further significant difference and the value was higher in group 2 than the group 3.P value was 0.016.

During pneumoperitoneum also the PaCO2 was significantly higher in group 1 than the other 2 groups.p value is 0.000. Even groups 2 and 3 varied significantly among themselves ie; group 2 showed a higher value than group 3 . p value is 0.004.

After deflation also the PaCO2 remained at higher level in group 1 than groups 2 and 3. p value is 0.000.

**ETCO2**

Measurements showed a significantly higher value of 33.5 mm of Hg in group 1 . (p value 0.000). Group 2 had a significantly higher value than

the group 3and the p value is 0.001.

During pneumoperitoneum also the values were significantly higher in group 1 than the groups 2 and 3 . p value is 0.000 . Group 2 showed a significantly higher value of 31.03 mm of Hg than group 3 and the p value is 0.030.

After deflation also group 1 had a higher value of 35.43mm of Hg than the groups 2 and 3 which had values of 29.36 mm of Hg and 29.166 mm of Hg respectively (p value is 0.000). Groups 2 and 3 did not show significant difference and the p value was 0.789.

**HCO3**

Bicarbonate levels measured before pneumoperitoneum did not show any significant difference among the three groups and the p value was 0.372.

During pneumoperitoneum also bicarbonate values did not show any statistically significant difference and the p value was 0.590. Values measured after disflation also showed no significant difference among the groups and the p value was 0.970.

**Pulse Rate**

Pre pneumoperitoneum values showed no significant difference among the groups and the p value is 0.143.

During pneumoperitoneum there was no significant change in the pulse rate in all the 3 groups. After deflation the values showed significant

increase in the study groups than the control group and the p value was 0.000. But this was not clinically significant.

## Peak inspiratory pressure

Prepneumoperitoneum values did not differ significantly between the groups and the p value was 0.976. During pneumoperitoneum the groups 2 and 3 showed a significantly higher values than the group 1 and the p value was 0.000.

But after deflation the values were not significantly different and the p value was 0.608. Our study shows that increasing the minute ventilation by 15 % in group2 and 30 % in group3 keeps the partial pressure of carbon dioxide and End tidal Carbon dioxide and pH within normal limits but it did not have significant changes in mean arterial pressure and heart rate .

## SUMMARY

We studied the effects of hyper ventilation on haemodynamics, partial pressure of carbondioxide and end tidal carbondioxide and acid base status of the patients undergoing laparoscopic cholecystectomy.

There were no significant differences between the groups in the demographic data.

There was no significant difference between the groups in the duration of surgery and duration of anaesthesia.

There was no significant difference in between the groups in the mean arterial blood pressure. But the heart rate was significantly higher in the control group than the study groups during pneumoperitoneum.

There was a statistically significant increase in pH in study groups when compared to control group .But this increase was not clinically significant .Because the study groups showed high normal values of pH whereas the control group showed the lower normal pH .

There was a significant decrease in Partial pressure of carbon dioxide and end tidal carbon dioxide in study groups when compared to the control groups during and after pneumoperitoneum.

There was no significant difference in the bicarbonate values.

# CONCLUSION

Carbon dioxide output increases during laparoscopic surgeries and increasing the minute ventilation by increasing the respiratory rate from 12 per minute to 14 per minute in laparoscopic surgeries produces a significant decrease in Partial pressure of carbon dioxide, End tidal carbon dioxide and pH towards the high normal range levels. When the rate was increased to 16 per minute the same changes were observed but this was not statistically significant from patients ventilated with a rate of 14 per minute. There was no significant difference in the bicarbonate values. There was no significant difference in between the groups in the mean arterial blood pressure. But the heart rate was significantly higher in the control group than the study groups during pneumoperitoneum.

PROFORMA

Group :

Name :

Age :

Sex :

Weight :

Type of surgery :

Duration of anaesthesia :

Duration of surgery :

| | MAP | PR | pH | PCO2 | ETCO2 | HCO3 |
|---|---|---|---|---|---|---|
| Pre Pneumo peritoneum | | | | | | |
| During pneumo peritoneum | | | | | | |
| Post pneumo peritoneum | | | | | | |

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